

A case of hypothyroidism presenting as dilated cardiomyopathy

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ABSTRACT

Thyroid disorders including hyperthyroidism and hypothyroidism may affect the cardiac function. In particular, inadequately treated hypothyroidism can cause diastolic and systolic dysfunction. Dilated cardiomyopathy, a condition that causes a decrease in the systolic function of the heart, is a rare manifestation of hypothyroidism, reversible with thyroid hormone therapy. We report a clinical case of reversible hypothyroidism induced dilated cardiomyopathy.

Keywords: Cardiomyopathy, Dilated cardiomyopathy, Hypothyroidism

1. INTRODUCTION

Dilated cardiomyopathy (DCM), the most common form of cardiomyopathy, is a cardiac muscle disorder defined by the presence of a dilated and poorly functioning left ventricle in the absence of abnormal loading conditions or extensive coronary artery disease (CAD), hypertension, heart valve disease and congenital heart disease. Patients with this condition generally have a poor prognosis due to a progressive and irreversible myocardial dysfunction. Nevertheless, with a detailed clinical history and a careful study, some rare treatable etiologies may be found such as endocrine disturbances (Elliott et al., 2008; Chaturvedi et al., 2020; Andhale et al. 2020). The thyroid gland is embryological and physiological related to the heart (Klein, 2015). Thyroid disorders, particularly hypothyroidism, have well established effects on the cardiovascular system (Klein & Ojamaa, 2001), although DCM seldom is the first manifestation. We report a clinical case of reversible hypothyroidism induced DCM. The short-term follow-up (2 weeks) and excellent echocardiographic documentation during follow-up represents an important contribution to clinical research of DCM's etiology.

2. CASE REPORT

A 39-year-old female presented to our clinic because of dyspnea. She noted that she has had shortness of breath on daily activities for one and a half months. The symptom was persistent and progressive. She also complained that she could not sleep well due to orthopnea and paroxysmal nocturnal dyspnea. Besides, both her lower limbs were swelling and she got oliguria without dysuria or changing urinary habits. She stated that she could not



tolerate cold and was constipated in recent months. She refused any other symptoms such as: fever, joint pain, chest pain, cough or loss of appetite. Her medical history included miscarriage at the 30th week of pregnancy and dilated cardiomyopathy (DCM) suspecting peripartum cardiomyopathy based on electrocardiograph (ECG), chest radiograph and echocardiography. She was prescribed beta blocker, angiotensin converting enzyme inhibitor (ACEI) and diuretics for one month. She found that the symptoms did not improve significantly. This led her to visit our clinic.

Physical examination

Patient appeared conscious, fatigue, puffy face with pale, cool and dry skin. She had a crowing voice and bilateral pedal edema, which was non pitting. Jugular venous pressure (JVP) was not raised. Her blood pressure was 88/52 mm Hg, pulse 69 bpm. The thyroid gland is not palpable. Her apex beat was in 6th intercostal space in the anterior axillary line. S1 and S2 were normal. There was no murmur, no crackles. There was no hepatomegaly, no ascites.

Workup

Chest X-rays showed enlarged cardiac silhouette (Figure 1). ECG sinus rhythm, with low voltage of limb leads (Figure 2). Her echocardiography was done which showed enlargement of the left ventricle (LV) and left atrium (Figure 3A) with severe LV global hypokinesis and ejection fraction Simpson biplane of 35%, GLS -10.4% (Figure 3B). There was moderate to severe mitral regurgitation, mild tricuspid regurgitation. Right atrium and right ventricle were normal with good right ventricle contractility. There was no pericardial effusion. Her renal functions were normal. Total cholesterol was 10.29 mmol/l, LDL 7.0 mmol/l and HDL 1.55 mmol/l. Her triglyceride levels were 2.1 mmol/l. Mild anemia with Hb 10.5g/dl, MCV 97.5 fL, MCH 31.8 pg and MCHC 32.6 g/dl. NT-proBNP was elevated 692 pg/ml.

Monitoring changes in voice and pedal edema, without increased JVP and hepatomegaly, she was consulted to do thyroid function test (TFT). She had severe hypothyroidism with TSH 81.147 μ U/mL, free T4 0.24 ng/dl. Thyroid ultrasound characterized by a diffuse low thyroid echogenicity associated with a reduced thyroid volume suggesting atrophic thyroiditis.

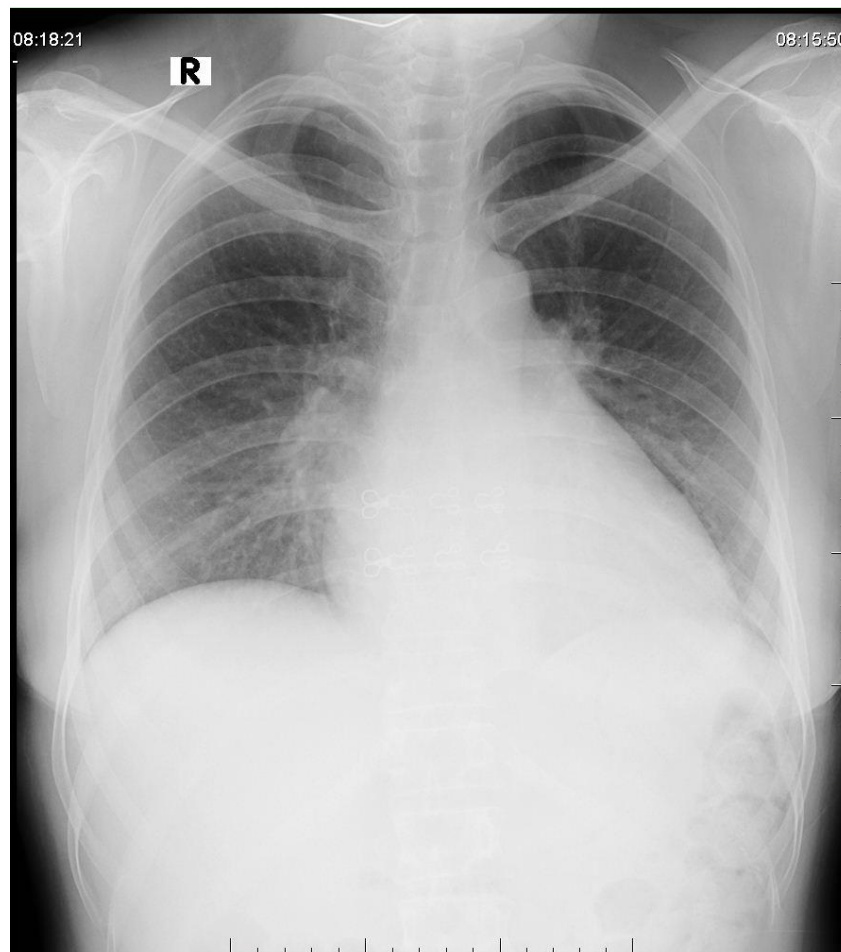


Figure 1 Chest X-rays showed enlarged cardiac silhouette

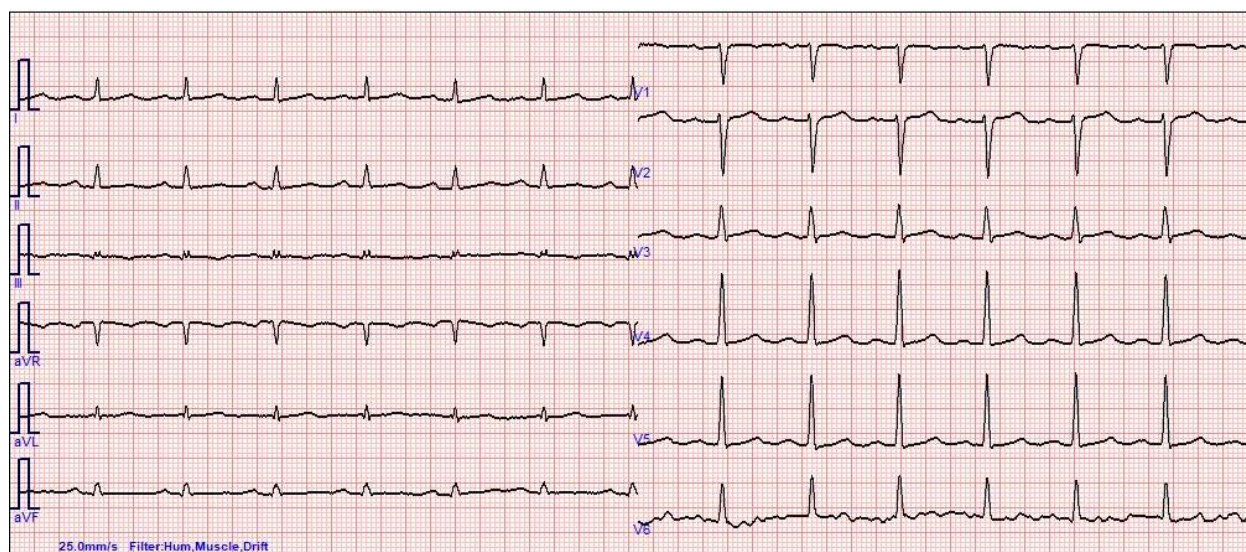


Figure 2 ECG normal sinus rhythm, with low voltage of limb leads

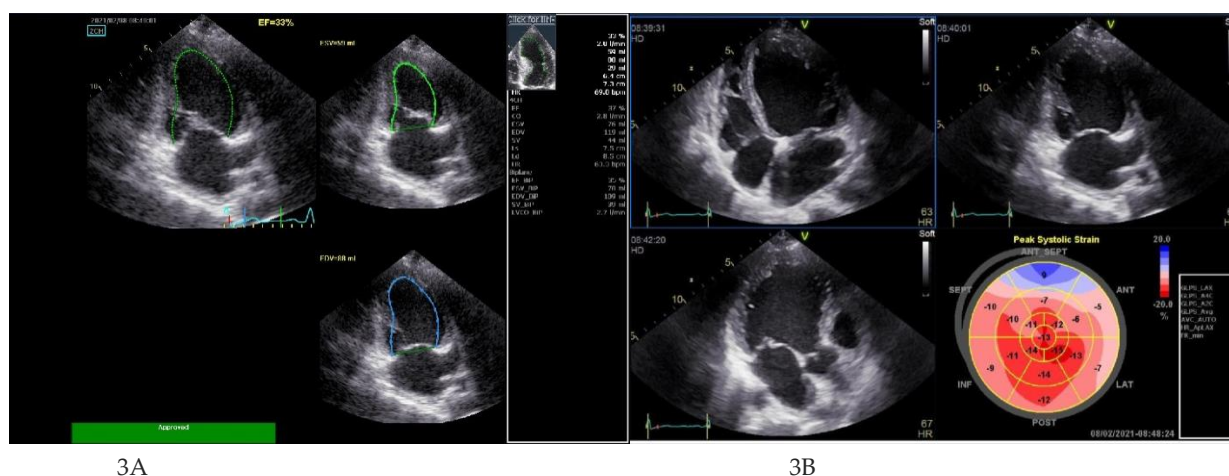


Figure 3A Enlargement of the left ventricle (LV) and left atrium. **3B.** Speckle tracking echocardiography showed decreased global longitudinal strain (GLS) -10.4%

Diagnosis and Management

Hypothyroidism-induced dilated cardiomyopathy. She was promptly initiated on levothyroxine 100 μ /day in addition to the therapy she was receiving for dilated cardiomyopathy including sacubitril/valsartan, spironolactone, furosemide.

3. DISCUSSION

The current report describes a patient with a typical congestive heart failure (HF) of recent onset. The evidence of a severe dilated left ventricular cavity with LVEF of 35% on TTE was consistent with the diagnosis of DCM. Other imaging diagnosis such as cardiac magnetic resonance (CMR) have high precision in the diagnosis of patients with DCM, displaying and quantifying functional abnormalities in DCM, showing ventricular volumes and LVEF and providing morphologic information (detecting myocardial scar, useful to characterize myocardial tissue) (Silva Marques & Pinto, 2015). Other than that, CMR has excellent reproducibility so it can be used for serial monitoring of cardiac function. However, widely available TTE remains the cornerstone in the algorithm for the diagnosis of patients with HF with reduced ejection fraction (Mc Murray et al., 2012). In the current case, lack of CMR could be pointed out as a limitation, as TTE was the main tool for the diagnosis and follow-up evaluation of DCM. In this young patient the absence of major cardiovascular risk factors led to a low probability of ischemic heart disease.

Patient had moderate to severe mitral regurgitation, however this abnormality occurred with varying frequency in dilated cardiomyopathy and it could not explain the reason why EF reduced severely without associated DCM. Congenital heart defects were easily excluded by the cardiac ultrasound evaluation. Other causes potentially related to transient DCM need to be excluded

was peripartum cardiomyopathy. According to diagnostic criteria of peripartum cardiomyopathy satisfy all of the following: 1. Development of heart failure in the last month of pregnancy or within the 5 months following delivery. 2. Absence of determinable etiology of heart failure. 3. Absence of demonstrable heart disease, prior to the diagnosis of heart failure. 4. Left ventricular ejection fraction < 45%, or fractional shortening <30%, or both (Hibbard, 1999). Although this patient had criteria 1, 3 and 4, she had hypothyroidism which can induce DCM. As we know, the diagnosis of peripartum cardiomyopathy is an exclusive diagnosis and if there is one of the causes of heart failure (such as hypothyroidism particularly in this case), then we must think about it first.

Thyroid gland functional disorders are relatively common in HF patients and thyroid function is highly recommended in order to detect reversible/treatable causes of HF, monitor treatment and establish a prognosis (McMurray et al., 2012). Through genomic and non-genomic mechanisms, biologically active thyroid hormones increase inotropism and chronotropism, decreases systemic vascular resistance and increases cardiac output (Klein, 2015; Klein & Ojamaa, 2001). The microcirculation of the myocardium also seems to be affected (Tang et al., 2005). Hyper and hypothyroidism can both produce changes in the heart and cardiovascular system (Klein, 2015; Klein & Ojamaa, 2001). In hypothyroidism, cardiac output may be reduced from 30% to 50% (Klein & Danzi, 2007). However, some cases of DCM have been described with regression of HF manifestations and reversal of dilated pattern after replacement hormonal treatment and correction of thyroid function. In fact, ESC guidelines mention hypothyroidism as a precipitant cause of acute HF (McMurray et al., 2012). In this case the treatment of thyroid abnormality with L-thyroxine was determinant for HF management and improvement in cardiac systolic function (Duntas, 2002).

Follow-up

Within 2 weeks of treating thyroxine, sacubitril/valsartan, spironolacton, furosemide, the patient started showing significant improvement in her symptoms of dyspnea and effort intolerance. A repeat echocardiographic examination after 2 weeks of replacement therapy revealed remarkable improvement in ejection fraction to 29% (Figure 4A) and improved GLS -12.1% (Figure 4B). There was a concurrent improvement in thyroid profile with the therapy (TSH 10.227 μ IU/ml, free T4 1.63 ng/dl).

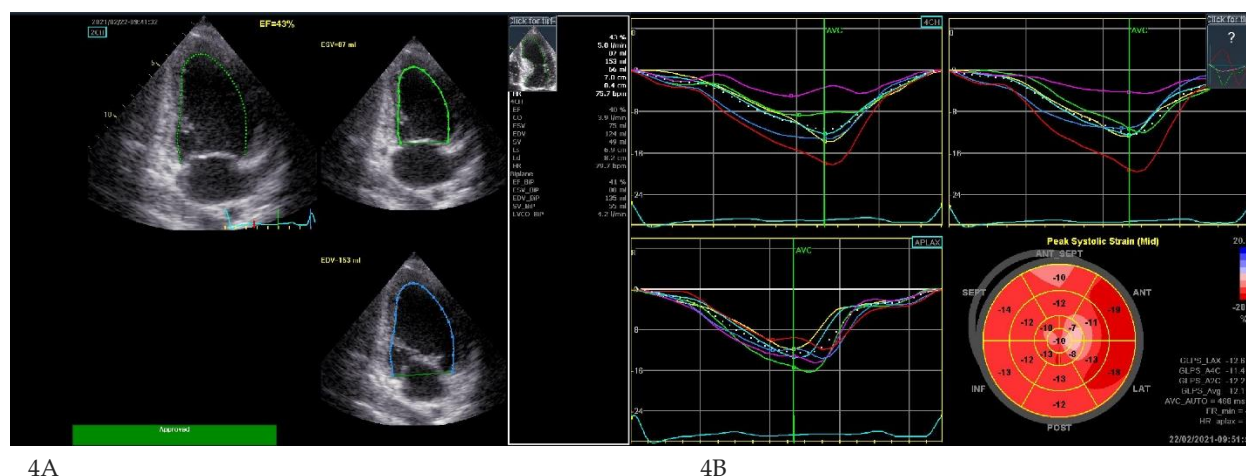


Figure 4 Echocardiography after 2 weeks. **4A.** EF Simpson biplane 41%. **4B.** Speckle tracking echocardiography showed improved (GLS) -12.1%

4. CONCLUSION

DCM is usually an idiopathic disease with progressive and an irreversible poor prognosis outcome. In contrast, in some cases, DCM can be secondary to various causes such as hypothyroidism and hormonal treatment with Levothyroxine can significantly improve myocardial function. Hence, thyroid function tests should be systematically performed in all patients with DCM in order to rule out a hypothyroidism.

Conflicts of Interest

The authors have no conflicts of interest that are directly relevant to the content of this clinico-pathological case.

Financial Resources

There are no financial resources to fund this study.

Informed consent

Informed consent was obtained from the patient.

Authors' Contributions

All the authors contributed equally to the case report

Data and materials availability

All data associated with this study are present in the paper.

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